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☐	L1	(corticotropin release inhibiting factor) or crif	25

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NEWS 25	DEC 22	IFIPAT/IFIUDB/IFICDB reloaded with new data and search fields

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FILE 'BIOSIS' ENTERED AT 13:39:35 ON 22 DEC 2003
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=> s (corticotropin release inhibiting factor) or crif
L1 32 (CORTICOTROPIN RELEASE INHIBITING FACTOR) OR CRIF

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L2 ANSWER 1 OF 24 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 2002:294010 BIOSIS
DN PREV200200294010
TI Anti-depressant effects of **corticotropin release inhibiting factor**.
AU Redei, Eva [Inventor]
CS ASSIGNEE: The Board of Trustees of Northwestern University
PI US 6372713 April 16, 2002
SO Official Gazette of the United States Patent and Trademark Office Patents, (Apr. 16, 2002) Vol. 1257, No. 3. http://www.uspto.gov/web/menu/patdata.html. e-file.
CODEN: OGUPE7. ISSN: 0098-1133.
DT Patent
LA English
ED Entered STN: 15 May 2002
Last Updated on STN: 15 May 2002
AB Methods and compositions for treatment of depression in animals are provided.

L2 ANSWER 2 OF 24 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 2002:198042 BIOSIS
DN PREV200200198042
TI **Corticotropin release inhibiting factor** and methods of using same.
AU Redei, Eva [Inventor]; Aird, Fraser [Inventor]
CS ASSIGNEE: Northwestern University; The Trustees of the University of Pennsylvania
PI US 6348571 February 19, 2002
SO Official Gazette of the United States Patent and Trademark Office Patents, (Feb. 19, 2002) Vol. 1255, No. 3. http://www.uspto.gov/web/menu/patdata.html. e-file.
CODEN: OGUPE7. ISSN: 0098-1133.
DT Patent
LA English
ED Entered STN: 13 Mar 2002
Last Updated on STN: 13 Mar 2002
AB The invention includes a substantially pure preparation of a **corticotropin release inhibiting factor** (**CRIF**) peptide having from three to twenty one or to twenty five contiguous amino acids contained within the amino acid

sequence positioned between the fourth and fifth TRH sequence on a prepro-TRH protein.

L2 ANSWER 3 OF 24 MEDLINE on STN DUPLICATE 1
AN 2002154911 MEDLINE
DN 21884261 PubMed ID: 11886697
TI Nocturnal secretion of TSH and ACTH in male patients with depression and healthy controls.
AU Peteranderl Christian; Antonijevic Irina A; Steiger Axel; Murck Harald; Held Katja; Frieboes Ralf-Michael; Uhr Martin; Schaaf Ludwig
CS Max Planck Institute of Psychiatry, Kraepelinstrasse 10, D-80804 Munich, Germany.
SO JOURNAL OF PSYCHIATRIC RESEARCH, (2002 May-Jun) 36 (3) 189-96. Journal code: 0376331. ISSN: 0022-3956.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200207
ED Entered STN: 20020312
Last Updated on STN: 20020709
Entered Medline: 20020708
AB Profound alterations of the hypothalamic-pituitary-thyroid (HPT) and the hypothalamic-pituitary-adrenal (HPA) systems at the hypophyseal level have been described in affective disorder. To precisely characterize the basal alterations of both axes during sleep, we simultaneously investigated sleep EEG and the secretion of thyrotropin, ACTH and cortisol in nine drug-free male patients with depression in comparison to 10 healthy age and sex matched controls. In depressed patients the nearly diametrical nocturnal secretion of thyrotropin and ACTH was disturbed by significantly blunted thyrotropin values (TSH AUC 51.96+/-5.68 vs. 87.23+/-13.63, P<0.05) and elevated ACTH values (ACTH AUC 1804+/-161 vs. 1538+/-130, P<0.05) compared to controls. Moreover, cross correlation analysis revealed a highly negative association of 0 lag between thyrotropin and ACTH and between thyrotropin and cortisol in the control sample, indicating a physiological nocturnal negative correlation of HPT and HPA system. In the patients sample these associations were weak and reached not statistical significance. Therefore, as a descriptive tool, the ratio TSH/ACTH revealed a significant group difference between controls and patients in the first half of the night (TSH/ACTH AUC 6.50+/-0.42 vs. 3.35+/-0.31, P<0.05). Sleep-EEG analysis showed a shortened REM latency, a decrease of stage 2 and an increase of awake time in the patients. Our data support the hypothesis that both hypophyseal hormones reflect a common dysregulation of both systems in depression probably due to impaired action of TRH-related **corticotropin-release-inhibiting-factor** (**CRIF**). The ratio TSH/ACTH might be a tool to characterize alterations of both the HPT and HPA axis in depression during the first half of the night.

L2 ANSWER 4 OF 24 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 2003:294943 BIOSIS
DN PREV200300294943
TI EVIDENCE FOR AN INVOLVEMENT OF THE HPA AXIS IN THE NEUROPROTECTIVE EFFECTS OF CRF ANTAGONISTS AFTER FOCAL ISCHEMIA IN THE RAT.
AU Mackay, K. B. [Reprint Author]; Stiefel, T. H. [Reprint Author]; Verge, G. M. [Reprint Author]; Naeve, G. S. [Reprint Author]; Foster, A. C. [Reprint Author]
CS Neurocrine Biosciences, San Diego, CA, USA
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 392.13. http://sfn.scholarone.com. cd-rom. Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
DT Conference; (Meeting)
Conference; (Meeting Poster)

Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 25 Jun 2003
Last Updated on STN: 25 Jun 2003
AB A potential role for hypothalamic-pituitary-adrenal (HPA) axis activation in the pathophysiology of stroke is suggested by the observation that ACTH and cortisol levels are elevated acutely in stroke patients, and correlate with outcome (Olsson et al., 1992). We have investigated the involvement of modulation of the HPA axis response in the genesis of ischemic damage after middle cerebral artery (MCA) occlusion in the rat. The left MCA was permanently occluded under halothane anesthesia and animals sacrificed 24 hrs later for quantitative histology. Administration of the mixed CRF1/CRF2 antagonist astressin (30 nmol/kg, i.v.) at the time of occlusion reduced the volume of total infarction by 30% ($P < 0.01$) relative to controls. There was no difference in ACTH levels between groups in the same animals, although the levels of corticosterone (CORT) were reduced by approximately 40% ($P < 0.05$) at 60-90 mins post-ischemia following astressin. Central administration of astressin (30 nmol/5mul; i.c.v) also reduced total infarct volume (by 24%; $P < 0.05$) when administered at the time of ischemia onset. Adrenalectomy (ADX) resulted in a 24% ($P < 0.05$) reduction in total infarct size relative to sham-operated controls, an effect that was eliminated upon treatment with CORT (5 mg; s.c.) 4 hrs prior to the ischemic insult. These results indicate that modulation of HPA axis activity can influence the degree of ischemic damage in an experimental model of stroke, and suggest that CRF receptor antagonists may have a novel profile of neuroprotective effects that include both central and peripheral (via the HPA axis) components.

L2 ANSWER 5 OF 24 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 2003:294944 BIOSIS
DN PREV200300294944
TI STRESS MEDIATES STROKE - INDUCED NEUROBEHAVIORAL DEFICITS IN P7B2 CUSHINGOID KNOCKOUT MICE.
AU Stahl, C. E. [Reprint Author]; Redei, E.; Borlongan, C. V.
CS Dept Internal Medicine, Dwight E. Eisenhower Army Medical Ctr, Fort Gordon, GA, USA
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 392.14. http://sfn.scholarone.com. cd-rom. Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
DT Conference; (Meeting)
Conference; (Meeting Poster)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 25 Jun 2003
Last Updated on STN: 25 Jun 2003
AB PPTRH 178-199 may act as an endogenous **corticotropin-release inhibiting factor** since its intravenous or intracerebral injections significantly reduced CORT and ACTH elevations produced by stress. Hypercorticolism is a hallmark of Cushing's disease, and a mouse model (7B2 knockout) of this disorder has been characterized. In the present study, we examined whether PPTRH 178-199 exert positive effects in 7B2 knockout mice exposed to stroke. Animals received daily systemic injections of PPTRH 178-199 or vehicle for 2 weeks, and monitored daily for appearance of cushingoid symptoms for 6 weeks. Blood samples were collected weekly for ACTH and CORT assays. On week 6, animals were subjected to stroke (via middle cerebral artery occlusion). Twenty four hours after stroke, animals were euthanized and brains were assayed for extent of cerebral infarction. Results revealed that PPTRH 178-199-treated 7B2 knockout mice significantly displayed less impairments in passive avoidance task and general spontaneous locomotor activity compared to vehicle-treated 7B2 knockout mice. In addition, treatment with PPTRH 178-199 significantly reduced cortical infarction, as well as ACTH and CORT levels in 7B2 knockout mice. These results suggest

that PPTRH 178-199 can reduce the stress in 7B2 knockout mice, thereby attenuating neurobehavioral deficits. Managing stress should be considered as vital therapeutic regimen for treating neurological disorders, such as stroke.

L2 ANSWER 6 OF 24 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:521696 BIOSIS
DN PREV200100521696
TI PPTRH 178-199 delays onset of cushingoid symptoms and prolongs survival of 7B2 knockout mice.
AU Borlongan, C. V. [Reprint author]; Coggiano, M. [Reprint author]; Snable, J.; Snable, G.; Vigersky, R.; Redei, E.; Stahl, C. E. [Reprint author]
CS Cell Neurobiol, NIDA, NIH, Baltimore, MD, USA
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 897. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 7 Nov 2001
Last Updated on STN: 23 Feb 2002
AB PPTRH 178-199 may act as an endogenous **corticotropin-release inhibiting factor** since its intravenous or intracerebral injections significantly reduced CORT and ACTH elevations produced by stress. Hypercorticolism is a hallmark of Cushing's disease, and a mouse model (7B2 knockout) of this disorder has been characterized. We treated 4 mice litters (bred from 7B2-/+) with either PPTRH 178-199 (200 ug/kg) (n=2 litters) or saline (n=2 litters) daily over 2 weeks, starting at PN1. In a blinded fashion, we monitored onset of cushingoid symptoms and survival, and collected blood samples weekly. The appearance of cushingoid symptoms (pale and severe bruising) in saline-treated -/- (n=7) was evident as early as PN4, whereas these symptoms only became evident at PN9 in peptide-treated -/- (n=8). Moreover, mortality in saline-treated -/- was noted between weeks 4 (n=4) and 8 (n=3), while the earliest death observed in peptide-treated -/- was at week 7 (n=3), with some surviving up to week 10 (n=3) and week 12 (n=2). No cushingoid symptoms were noted in -/+ (n=9) or +/+ (n=11), and they appeared normal throughout the 20-week study. ANOVA revealed significant treatment effects on ACTH and CORT levels, with posthoc tests indicating that peptide-treated -/- did not differ from +/+ up to week 3 (27.8 ug/dl CORT; 23 pg/ml ACTH), while saline-treated -/- had significant high levels of these hormones from week 1 (98 ug/dl CORT; 142 pg/ml ACTH). These results suggest that PPTRH 178-199 has therapeutic effects in an animal model of Cushing's disease.

L2 ANSWER 7 OF 24 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 2000:425938 BIOSIS
DN PREV200000425938
TI **Corticotropin release inhibiting factor** and methods of using same for treating behavioral symptoms in an anxiety disorder.
AU Redei, Ev [Inventor, Reprint author]; Aird, Fraser [Inventor]; Rittenhouse, Peter A. [Inventor]; McGivern, Robert F. [Inventor]
CS Chicago, IL, USA
ASSIGNEE: Pennsylvania, Trustees of the University of, The, Philadelphia, PA, USA
PI US 6039956 March 21, 2000
SO Official Gazette of the United States Patent and Trademark Office Patents, (Mar. 21, 2000) Vol. 1232, No. 3. e-file.
CODEN: OGUPE7. ISSN: 0098-1133.
DT Patent
LA English
ED Entered STN: 4 Oct 2000

Last Updated on STN: 10 Jan 2002

AB The invention includes a substantially pure preparation of a peptide having **corticotropin release inhibiting factor** (**CRIF**) activity comprising at least three contiguous amino acids contained within the amino acid sequence positioned between the fourth and fifth thyrotropin releasing hormone (TRH) sequence on a prepro-TRH protein. The **CRIF** peptide further comprises the fourth uncleaved TRH portion of prepro-TRH positioned at the amino terminus of **CRIF**. Compositions, methods of diagnosis and methods of treating **CRIF** related diseases are also included in the invention.

L2 ANSWER 8 OF 24 MEDLINE on STN DUPLICATE 2
AN 2001025914 MEDLINE
DN 20461108 PubMed ID: 11004712
TI Role of the hypothalamic pituitary adrenal axis in the control of the response to stress and infection.
AU McCann S M; Antunes-Rodrigues J; Franci C R; Anselmo-Franci J A; Karanth S; Rettori V
CS Pennington Biomedical Research Center (LSU), Baton Rouge, LA 70808-4124, USA.. mccansm@mhs.pbrc.edu
NC DK43900 (NIDDK)
MH51853 (NIMH)
SO BRAZILIAN JOURNAL OF MEDICAL AND BIOLOGICAL RESEARCH, (2000 Oct) 33 (10) 1121-31. Ref: 47
Journal code: 8112917. ISSN: 0100-879X.
CY Brazil
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200011
ED Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001116

AB The release of adrenocorticotropin (ACTH) from the corticotrophs is controlled principally by vasopressin and corticotropin-releasing hormone (CRH). Oxytocin may augment the release of ACTH under certain conditions, whereas atrial natriuretic peptide acts as a **corticotropin release-inhibiting factor** to inhibit ACTH release by direct action on the pituitary. Glucocorticoids act on their receptors within the hypothalamus and anterior pituitary gland to suppress the release of vasopressin and CRH and the release of ACTH in response to these neuropeptides. CRH neurons in the paraventricular nucleus also project to the cerebral cortex and subcortical regions and to the locus ceruleus (LC) in the brain stem. Cortical influences via the limbic system and possibly the LC augment CRH release during emotional stress, whereas peripheral input by pain and other sensory impulses to the LC causes stimulation of the noradrenergic neurons located there that project their axons to the CRH neurons stimulating them by alpha-adrenergic receptors. A muscarinic cholinergic receptor is interposed between the alpha-receptors and nitric oxidergic interneurons which release nitric oxide that activates CRH release by activation of cyclic guanosine monophosphate, cyclooxygenase, lipoxygenase and epoxygenase. Vasopressin release during stress may be similarly mediated. Vasopressin augments the release of CRH from the hypothalamus and also augments the action of CRH on the pituitary. CRH exerts a positive ultrashort loop feedback to stimulate its own release during stress, possibly by stimulating the LC noradrenergic neurons whose axons project to the paraventricular nucleus to augment the release of CRH.

L2 ANSWER 9 OF 24 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:135235 BIOSIS

DN PREV200100135235
TI Inhibition of the corticotropin-releasing hormone activity in the hypothalamus of the restrained rats by prepro-TRH 178-199.
AU Fukagawa, T. [Reprint author]; Fukagawa, K.; Gotoh, K.; Noguchi, H.; Yoshimatsu, H.; Sakata, T.
CS Oita Medical University, Oita, Japan
SO Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-780.7. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 04-09, 2000. Society for Neuroscience.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 14 Mar 2001
Last Updated on STN: 15 Feb 2002
AB In 1995, Redei et al. reported that the preproTRH 178-199 (pTRH) suppressed the increase of the plasma ACTH in the restrained rats as corticotropin-releasing hormone (CRH) inhibitory factor (**CRIF**). There are still controversies whether pTRH can directly stimulate ACTH secretion of the cultured pituitary cells. It is known that restraint stress cause the activation of the rat hypothalamic histamine neuron. The aim of this study was to clarify whether pTRH could affect the CRH concentration in the hypothalamus of the restrained rats and rats with central administration of histamine. 1)Before and after histamine (270nmol) 3rd ventricular infusion (icv), the blood of the Sprague-Dawly rats with pTRH (6mug/kg)or saline pretreatment was collected and plasma ACTH concentration was measured.2)The brains of the non-restrained rats (NR), restrained rats (Rest) and restrained rats with the pretreatment of pTRH (6mug/kg,icv)(R-pTRH) were removed and frozen. The PVN, the ventromedial nucleus(VMN), the lateral hypothalamus (LH) and the amygdala(AG) ware collected from the frozen brain sections. CRH concentration was measured from the extracts of each hypothalamic nucleus. The plasma ACTH concentration increased 4 fold after histamine icv and the pretreatment of the pTRH attenuated this ACTH increment by histamine icv($P<0.05$).The CRH concentration in the PVN and the VMH of the Rest increased compared to those of the NR ($p<0.05$). There were no differences in the CRH concentration between the R-pTRH and the NR. These results demonstrated that p-TRH may directly inhibit the CRH activity in the PVN and the VMH of the restrained rats as **CRIF**.

L2 ANSWER 10 OF 24 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 2000:147273 BIOSIS
DN PREV200000147273
TI **Corticotropin release-inhibiting factor** reduces stress- and stroke-induced behaviors and brain infarction.
AU Stahl, C. E. [Reprint author]; Lin, S. Z. [Reprint author]; Wang, Y.; Chiang, Y. H. [Reprint author]; Redei, E.; Borlongan, C. V.
CS Department of Neurosurgery, National Defense Medical Center, Taipei, Taiwan
SO Society for Neuroscience Abstracts, (1999) Vol. 25, No. 1-2, pp. 1851. print.
Meeting Info.: 29th Annual Meeting of the Society for Neuroscience. Miami Beach, Florida, USA. October 23-28, 1999. Society for Neuroscience.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 19 Apr 2000
Last Updated on STN: 4 Jan 2002

L2 ANSWER 11 OF 24 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 2002:127176 BIOSIS

DN PREV200200127176
TI **Corticotropin release inhibiting factor** and methods of using same.
AU Redei, E. [Inventor]; Aird, F. [Inventor]
CS Philadelphia, Pa., USA
ASSIGNEE: THE TRUSTEES OF THE UNIVERSITY OF PENNSYLVANIA
PI US 5830866 Nov. 3, 1998
SO Official Gazette of the United States Patent and Trademark Office Patents, (Nov. 3, 1998) Vol. 1216, No. 1, pp. 587. print.
CODEN: OGUPE7. ISSN: 0098-1133.
DT Patent
LA English
ED Entered STN: 30 Jan 2002
Last Updated on STN: 26 Feb 2002

L2 ANSWER 12 OF 24 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 1998:374593 BIOSIS
DN PREV199800374593
TI A novel endogenous **corticotropin release inhibiting factor**.
AU Redei, Eva [Reprint author]; Rittenhouse, Peter A.; Revskoy, Sergei [Reprint author]; McGivern, Robert F.; Aird, Fraser [Reprint author]
CS Asher Cent., Dep. Psychiatry Behavioral Sci., Northwestern Univ. Medical Sch., 303 East Chicago Ave., Ward Bldg., 9-142, Chicago, IL 60611, USA
SO McCann, S. M. [Editor]; Liptin, J. M. [Editor]; Sternberg, E. M. [Editor]; Chrousos, G. P. [Editor]; Gold, P. W. [Editor]; Smith, C. C. [Editor]. Ann. N. Y. Acad. Sci., (1998) pp. 456-469. Annals of the New York Academy of Sciences; Neuroimmunomodulation: Molecular aspects, integrative systems, and clinical advances. print.
Publisher: New York Academy of Sciences, 2 East 63rd Street, New York, New York 10021, USA. Series: Annals of the New York Academy of Sciences.
Meeting Info.: Third Congress of the International Society of NeuroImmunoModulation (ISNIM). Bethesda, Maryland, USA. November 13-15, 1996. International Society for NeuroImmunoModulation.
CODEN: ANYAA9. ISSN: 0077-8923. ISBN: 1-57331-072-7 (cloth), 1-57331-073-5 (paper).
DT Book
Conference; (Meeting)
Book; (Book Chapter)
Conference; (Meeting Paper)
LA English
ED Entered STN: 2 Sep 1998
Last Updated on STN: 21 Oct 1998

L2 ANSWER 13 OF 24 MEDLINE on STN
AN 1998292917 MEDLINE
DN 98292917 PubMed ID: 9629272
TI A novel endogenous **corticotropin release inhibiting factor**.
AU Redei E; Rittenhouse P A; Revskoy S; McGivern R F; Aird F
CS Department of Pharmacology, University of Pennsylvania, Philadelphia 19104, USA.. e-redei@nwu.edu
SO ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, (1998 May 1) 840 456-69. Ref: 95
Journal code: 7506858. ISSN: 0077-8923.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199807
ED Entered STN: 19980716
Last Updated on STN: 19980716

Entered Medline: 19980709

AB ACTH is the major regulator of the body's adaptive response to stress and the physiological stimulus for glucocorticoid secretion. A hypothalamic **corticotropin release inhibiting factor** (**CRIF**) that inhibits ACTH synthesis and secretion has long been postulated but was not characterized until recently. We have recently identified a 22 amino acid peptide, prepro-thyrotropin releasing hormone (TRH) 178-199 that inhibits basal and stimulated ACTH synthesis and secretion in vitro and stress-induced ACTH secretion in vivo. Prepro-TRH 178-199 is abundant in several brain regions, including the external zone of the median eminence, where its concentration changes in response to stress. We propose that this peptide is a physiological regulator of ACTH production: an endogenous **CRIF**. Because prepro-TRH 178-199 is encoded within the same precursor as TRH, its expression is likely to be negatively regulated by thyroid hormones leading to changes in endogenous glucocorticoid levels. Streptococcal cell wall (SCW)-induced inflammation, a model of rheumatoid arthritis (RA), was alleviated after long-term thyroxine treatment. Inversely, a hypothyroid milieu led to decreased basal hypothalamic-pituitary-adrenal activity, but increased expression of IL-1 beta and MIP-1 alpha, specific markers for RA in humans. These results suggest that this putative **CRIF** may be an important component in the development of RA and that regulation of prepro TRH may be highly relevant to the development of other autoimmune diseases that are also exacerbated by low endogenous glucocorticoid levels.

L2 ANSWER 14 OF 24 MEDLINE on STN DUPLICATE 3
AN 97313552 MEDLINE
DN 97313552 PubMed ID: 9169546
TI Inhibition of stress-induced neuroendocrine and behavioral responses in the rat by prepro-thyrotropin-releasing hormone 178-199.
AU McGivern R F; Rittenhouse P; Aird F; Van de Kar L D; Redei E
CS Department of Psychology, San Diego State University, San Diego, California 92182, USA.
NC AA06478 (NIAAA)
SO JOURNAL OF NEUROSCIENCE, (1997 Jun 15) 17 (12) 4886-94.
Journal code: 8102140. ISSN: 0270-6474.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Space Life Sciences
EM 199706
ED Entered STN: 19970716
Last Updated on STN: 19970716
Entered Medline: 19970630

AB A **corticotropin release-inhibiting factor** (**CRIF**) in brain has been postulated for several decades, based on increased plasma levels of ACTH and corticosterone after hypothalamic-pituitary disconnection. Recent in vitro studies indicate that prepro-TRH178-199 may function as an endogenous **CRIF**, prompting us to examine stress-related neuroendocrine and behavioral responses after in vivo administration to the adult male rat. Animals that were administered prepro-TRH178-199 intravenously 5 min before restraint stress exhibited a significant attenuation of stress-induced elevations of ACTH, corticosterone, and prolactin, as compared with controls infused with vehicle, whereas thyroid-stimulating hormone (TSH) secretion was not changed. In behavioral studies of stress responsiveness, either the vehicle or prepro-TRH178-199 was administered intracerebroventricularly (ICV) 5 min before testing. In the open field, prepro-TRH178-199 significantly increased grooming, locomotor activity, rearing, and sniffing behaviors. In the light/dark box, it significantly increased the time animals spent in the light compartment and increased the number of crossings between the light/dark compartments. In the plus maze, the peptide significantly increased the amount of time animals spent

in the open arms. The same dose of peptide, administered ICV, had no effect on peripheral hormone release in response to restraint stress. Overall, these results support a role for prepro-TRH178-199 in the inhibition of the neuroendocrine responses to stress at the level of the pituitary and indicate that it has central modulatory influences over stress-related behaviors.

L2 ANSWER 15 OF 24 MEDLINE on STN DUPLICATE 4
AN 1998148978 MEDLINE
DN 98148978 PubMed ID: 9487998
TI Residual pituitary function after transsphenoidal hypophysectomy in dogs with pituitary-dependent hyperadrenocorticism.
AU Meij B P; Mol J A; Bevers M M; Rijnberk A
CS Department of Clinical Sciences of Companion Animals, Faculty of Veterinary Medicine, Utrecht University, The Netherlands.
SO JOURNAL OF ENDOCRINOLOGY, (1997 Dec) 155 (3) 531-9.
Journal code: 0375363. ISSN: 0022-0795.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199803
ED Entered STN: 19980319
Last Updated on STN: 19980319
Entered Medline: 19980309
AB Pituitary function was assessed before and after transsphenoidal hypophysectomy in 39 dogs with pituitary-dependent hyperadrenocorticism (PDH). Anterior pituitary function was investigated using combined administration of four hypophysiotropic releasing hormones (corticotropin-releasing hormone (CRH), GHRH, GnRH, and TRH) with measurements of ACTH, cortisol, GH, LH, prolactin (PRL), and TSH Pars intermedia function was assessed by measurements of basal plasma alpha-MSH concentrations and adrenocortical function by baseline urinary corticoid/creatinine ratios. At eight weeks after hypophysectomy basal plasma ACTH, cortisol, GH, LH, PRL, and TSH concentrations were significantly lower than before surgery. In seven dogs with elevated alpha-MSH concentrations, the values returned to the normal level after surgery. In the combined anterior pituitary function test there were no plasma GH, LH, PRL, and TSH responses to stimulation, whereas plasma ACTH and cortisol responses were small but significant. Remission of hyperadrenocorticism was obtained in 35 dogs and recurrences occurred in 3 of these within 16 months postoperatively. At 8 weeks after hypophysectomy, these 3 dogs were not discernible, with respect to residual pituitary and adrenocortical function, from the 32 dogs with persisting remission. Urinary corticoid/creatinine ratios in the latter group of dogs did not increase during 22 months after hypophysectomy. In contrast to the presurgical findings, at 8 weeks after hypophysectomy there were significant positive correlations between baseline urinary corticoid/creatinine ratios and basal levels and responses for ACTH, indicating return to normal function of the pituitary-adrenocortical axis. It is concluded that among the adenohypophyseal cells present in the sella turcica after hypophysectomy, the corticotropes have a distinct behavior. Much more so than the other cell types, the unaffected corticotropes tend to remain functional, or a repressed reserve fraction of corticotropes may become functional. This may be due to the removal of the hypothalamic influence of a postulated **corticotropin-release inhibiting factor** or a diminished inhibitory influence of a postulated paracrine factor. The corticotropes may maintain normocorticism, but may also lead to mild recurrence after relatively long periods of remission.

L2 ANSWER 16 OF 24 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 1997:467235 BIOSIS
DN PREV199799766438

TI Localization of prepro-TRH-178-199, a **corticotropin release inhibiting factor**, in the brain of the rat.
AU Handa, R. J. [Reprint author]; McGivern, R. F.; Redei, E. E.
CS Dep. Cell Biol. Neurobiol. and Anat., Loyola Univ. Med. Cent., Maywood, IL 60153, USA
SO Society for Neuroscience Abstracts, (1997) Vol. 23, No. 1-2, pp. 119.
Meeting Info.: 27th Annual Meeting of the Society for Neuroscience, Part 1. New Orleans, Louisiana, USA. October 25-30, 1997.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LA English
ED Entered STN: 4 Nov 1997
Last Updated on STN: 10 Dec 1997

L2 ANSWER 17 OF 24 MEDLINE on STN DUPLICATE 5
AN 96198825 MEDLINE
DN 96198825 PubMed ID: 8612564
TI Preprothyrotropin-releasing hormone-(178-199) does not inhibit corticotropin release.
AU Nicholson W E; Orth D N
CS Department of Medicine, Vanderbilt University Medical Center, Nashville, Tennessee 37232-6303, USA.
SO ENDOCRINOLOGY, (1996 May) 137 (5) 2171-4.
Journal code: 0375040. ISSN: 0013-7227.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199606
ED Entered STN: 19960613
Last Updated on STN: 19960613
Entered Medline: 19960605
AB Pituitary ACTH synthesis and secretion are positively regulated by hypothalamic factors and negatively regulated by adrenal corticosteroids. Negative hypothalamic regulation of pituitary ACTH synthesis and secretion has been postulated, but not proved. The search for a hypothalamic **corticotropin release-inhibiting factor** has recently focused on peptides derived from the prepro-TRH precursor of TRH. One of the peptides, prepro-TRH-(178-199), was reported to suppress basal and stimulated ACTH release. We examined the effects of prepro-TRH-(178-199) alone and in combination with CRH and corticosterone, two known physiologic regulators of ACTH secretion. Prepro-TRH-(178-199) had no effect on basal, stimulated, or attenuated ACTH release from cells that responded normally to CRH and/or corticosterone. These results indicate that prepro-TRH-(178-199) is not a **corticotropin release-inhibiting factor.**

L2 ANSWER 18 OF 24 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 1996:552300 BIOSIS
DN PREV199699274656
TI Restraint stress produces region specific changes in rat brain prepro-TRH 178-199, a proposed novel **corticotropin release inhibiting factor.**
AU Rittenhouse, P. A. [Reprint author]; Zorrilla, E. P. [Reprint author]; McGivern, R. F.; Redei, E. [Reprint author]
CS Dep. Psychiatry, Univ. Pennsylvania, Philadelphia, PA 19104, USA
SO Society for Neuroscience Abstracts, (1996) Vol. 22, No. 1-3, pp. 1341.
Meeting Info.: 26th Annual Meeting of the Society for Neuroscience. Washington, D.C., USA. November 16-21, 1996.
ISSN: 0190-5295.

DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LA English
ED Entered STN: 13 Dec 1996
Last Updated on STN: 13 Dec 1996

L2 ANSWER 19 OF 24 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 1996:490108 BIOSIS
DN PREV199699212464
TI AtT20 cells transfected with the rat prepro-TRH cDNA exhibit increased sensitivity to ACTH suppression by dexamethasone: Proposed role of **corticotropin release inhibiting factor** (**CRIF**).
AU Revskoy, S.; Aird, F.; Whybrow, P.; Redei, E.
CS Lab. Neuroendocrinology, Dep. Psychiatry, University Pennsylvania, Philadelphia, PA 19104, USA
SO Society for Neuroscience Abstracts, (1996) Vol. 22, No. 1-3, pp. 843.
Meeting Info.: 26th Annual Meeting of the Society for Neuroscience. Washington, D.C., USA. November 16-21, 1996.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LA English
ED Entered STN: 4 Nov 1996
Last Updated on STN: 10 Dec 1996

L2 ANSWER 20 OF 24 MEDLINE on STN DUPLICATE 6
AN 95354608 MEDLINE
DN 95354608 PubMed ID: 7628393
TI **Corticotropin release-inhibiting factor** is preprothyrotropin-releasing hormone-(178-199).
AU Redei E; Hilderbrand H; Aird F
CS Department of Pharmacology, University of Pennsylvania, Philadelphia 19104, USA.
SO ENDOCRINOLOGY, (1995 Aug) 136 (8) 3557-63.
Journal code: 0375040. ISSN: 0013-7227.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199509
ED Entered STN: 19950921
Last Updated on STN: 19970203
Entered Medline: 19950901
AB ACTH is the major regulator of the body's adaptive response to stress and the physiological stimulus for glucocorticoid secretion. In addition to the known negative feedback regulation of ACTH by glucocorticoids, a hypothalamic **corticotropin release-inhibiting factor** (**CRIF**) that inhibits ACTH synthesis and secretion has been postulated, but not identified. We previously reported that transfection of prepro-TRH complementary DNA into the mouse anterior pituitary tumor cell line AtT-20 results in inhibition of basal and corticotropin-releasing hormone (CRH)-stimulated ACTH synthesis and secretion, suggesting that one or more of the cryptic peptides encoded within the prepro-TRH precursor has **CRIF** activity. To narrow the choice of peptides responsible for **CRIF** activity, we first deleted specific sequences within the prepro-TRH complementary DNA and transfected these constructs into AtT-20 cells. Deletion of sequences encoding amino acids 119-229 resulted in the loss of **CRIF** activity. Of the peptides encoded within this region, prepro-TRH-(178-199), a 22-amino acid peptide, inhibited basal and CRH-stimulated ACTH synthesis and secretion in cultured primary anterior

pituitary cells. As this peptide is processed from prepro-TRH in vivo, is found in the external zone of the median eminence, and is secreted from hypothalamic slices in vitro, prepro-TRH-(178-199) fulfills the criteria for a physiological **CRIF**. The significance of TRH and **CRIF** sharing a common precursor opens new areas of research in the integrated regulation of pituitary-adrenal and pituitary-thyroid functions.

L2 ANSWER 21 OF 24 MEDLINE on STN DUPLICATE 7
AN 95203251 MEDLINE
DN 95203251 PubMed ID: 7895696
TI **Corticotropin release inhibiting factor** is encoded within prepro-TRH.
AU Redei E; Hilderbrand H; Aird F
CS Department of Pharmacology, University of Pennsylvania, Philadelphia 19104.
SO ENDOCRINOLOGY, (1995 Apr) 136 (4) 1813-6.
Journal code: 0375040. ISSN: 0013-7227.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199504
ED Entered STN: 19950504
Last Updated on STN: 19970203
Entered Medline: 19950425
AB Corticotropin synthesis and secretion is under negative feedback regulation by glucocorticoids. In addition a hypothalamic factor inhibiting ACTH synthesis and secretion, named **corticotropin release inhibiting factor** (**CRIF**), has been postulated but not identified. Here we report that transient transfection of a rat prepro-TRH cDNA into the mouse anterior pituitary tumor cell line AtT-20 inhibits the synthesis and secretion of both basal and CRH-stimulated ACTH. Thus, **CRIF** appears to be encoded by the same precursor as TRH, suggesting a coordinated regulation of pituitary-adrenal and pituitary-thyroid functions.

L2 ANSWER 22 OF 24 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 1995:162999 BIOSIS
DN PREV199598177299
TI Delta-sleep-inducing peptide does not affect CRH and meal-induced ACTH and cortisol secretion.
AU Spaeth-Schwalbe, Ernst [Reprint author]; Schaefer, Arno; Uthgenannt, Dirk; Born, Jan; Fehm, Horst Lorenz
CS Universitaetsklinik Ulm, Med. Klinik, Innere III, Robert-Koch-Strasse 8, D-89081 Ulm, Germany
SO Psychoneuroendocrinology, (1995) Vol. 20, No. 3, pp. 231-237.
CODEN: PSYCDE. ISSN: 0306-4530.
DT Article
LA English
ED Entered STN: 11 Apr 1995
Last Updated on STN: 12 Apr 1995
AB Besides sleep-promoting properties, delta-sleep-inducing peptide (DSIP) has been reported to act as a **corticotropin-release inhibiting factor** in vitro and in vivo. We examined, first, the influence of DSIP on ACTH and cortisol release following stimulation with human corticotropin-releasing hormone (h-CRH; 1.0 mu-g/kg body weight, and 0.5 mu-g/kg body weight, respectively) in healthy young men (n = 5 in each condition). DSIP (total doses of 3 and 4 mg, respectively, vs. placebo) was infused intravenously between 30 min prior to and 90 min after CRH injections. Responses of ACTH and cortisol were almost identical during and after infusion of DSIP and placebo. In a second experiment, the influence of DSIP (4 mg, also administered as intravenous infusion) on meal-related ACTH and cortisol secretion was

studied in another 10 men. Meal-related midday surge of ACTH and cortisol was also not affected by DSIP. Our data do not support an inhibitory role of DSIP on ACTH and cortisol secretion in man.

L2 ANSWER 23 OF 24 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 1991:69120 BIOSIS
DN PREV199191037780; BA91:37780
TI ATRIOPEPTIN INHIBITS STIMULATED SECRETION OF ACTH IN RATS EVIDENCE FOR A PITUITARY SITE OF ACTION.
AU KOVACS K J [Reprint author]; ANTONI F A
CS MRC BRAIN METABOLISM UNIT, 1 GEORGE SQUARE, EDINBURGH EH8 9JZ, UK
SO Endocrinology, (1990) Vol. 127, No. 6, pp. 3003-3008.
CODEN: ENDOAO. ISSN: 0013-7227.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 29 Jan 1991
Last Updated on STN: 30 Jan 1991
AB The aim of this study was to resolve previous controversies regarding the effect of atriopeptin on the secretion of ACTH in vivo. Male Wistar rats were used throughout. The animals were subjected to lesioning of the hypothalamic paraventricular nucleus (PVN) or sham operation and implanted with indwelling jugular cannulae 5 days later for blood sampling and drug infusion. Two days after the insertion of the cannulae the animals were treated with saline or 103-126 amino acid residue atriopeptin iv: a bolus injection was given (200 or 40 pmol/rat) followed by an infusion (40 or 8 pmol/min) which was maintained for the entire duration of the experiment (70 min). Ten minutes after the bolus of atriopeptin the animals received iv a combination of 1 pmol 41-residue CRF and 10 pmol arginine vasopressin (CRF/AVP) to stimulate ACTH secretion. Serial blood samples (0.1 ml) were obtained at -10 min and immediately before the injection of CRF/AVP and at 5, 10, 20, 30, and 60 min afterwards. Plasma ACTH concentration was measured by RIA. In sham-operated rats CRF/AVP caused a 4-fold increase in plasma ACTH which peaked at 5 min and returned to baseline by 60 min. In sham-operated rats the higher dose of atriopeptin (200 pmol bolus, 40 pmol/min infusion) did not alter the effect of the stimulus between 5 and 30 min, and augmented plasma ACTH at 60 min. The smaller dose of atriopeptin reduced plasma ACTH at 10 and 20 min by 54% and 48%, respectively, and also decreased by 48% the net amount of ACTH released over 30 min in response to CRF/AVP. When given alone, the higher dose of atriopeptin caused a persistent (60 min) 10-13% reduction of mean arterial blood pressure, while the lower dose decreased blood pressure by about 9% for less than 10 min. In parallel, the higher dose of atriopeptin increased plasma ACTH concentration while the lower dose produced no change. In PVN-lesioned rats the CRF/AVP induced ACTH response was similar to that seen in sham-operated controls. Only the higher dose of atriopeptin was tested, and this markedly reduced CRF/AVP stimulated ACTH secretion at 5-60 min after CRF/AVP. Given alone, atriopeptin had no marked effect on plasma ACTH in PVN-lesioned rats, while its hypotensive action was similar to that in sham-operated animals. It is concluded that: 1) Atriopeptin inhibits stimulated ACTH secretion in rats; 2) High systemic doses of atriopeptin that cause persistent hypotension release endogenous ACTH-releasing factors of paraventricular nucleus origin, and this may mask an inhibitory action of the peptide at the pituitary level; 3) Atriopeptin may function as a hypothalamic **corticotropin release-inhibiting factor**.

L2 ANSWER 24 OF 24 MEDLINE on STN DUPLICATE 8
AN 82267111 MEDLINE
DN 82267111 PubMed ID: 7108215
TI The antigens of pigeon breeder's disease. VII. Isoelectric focusing studies on unfractionated pigeon dropping extract.
AU McCormick D J; Fredricks W W; Tebo T H; Calvanico N J
NC HL 15389 (NHLBI)

SO JOURNAL OF IMMUNOLOGY, (1982 Oct) 129 (4) 1493-8.
Journal code: 2985117R. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 198210
ED Entered STN: 19900317
Last Updated on STN: 19970203
Entered Medline: 19821029
AB Isoelectric focusing (IEF) studies on pigeon dropping extracts (PDE) revealed that it is a complex mixture of components that are acidic in nature. Chromatographically purified antigens PDEB, PDE1, and PDE3 showed multiple bands in IEF, indicating a microheterogeneity of these components, with peak concentrations focusing at pH 6.1, 4.6, and 3.8, respectively. The isoelectric points are compatible with the chromatographic behavior of these antigens on DEAE-cellulose. Crossed immunoelectrofocusing (**CRIF**) resolved PDE into seven precipitin lines with rabbit antiserum in a pH gradient from 3.5 to 9.5, and into nine precipitable components in a pH gradient from 2.5 to 7.0. The complexity of this antigen source appears to reside in the heterogeneity of immunologically related antigens. A comparison of pigeon breeder's sera by **CRIF** of PDE revealed a qualitative difference in precipitation patterns obtained with symptomatic and asymptomatic individuals. Sera from all (eight out of eight) of the symptomatic breeders tested precipitated an unidentified component of PDE, whereas none (zero out of four) of the sera from asymptomatic breeders detected this antigen. These results suggest that **CRIF** of PDE is useful as a diagnostic tool and that some specific component of PDE may be involved in the pathogenesis of the disease.

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